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THE EFFECT OF ALCOHOL AND AMPHETAMINE UPON BENDER-GESTALT REPRODUCTIONS

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF ARTS

DEPARTMENT OF PSYCHOLOGY

BY

NEIL JEFFREY GAMBLE

EDMONTON, ALBERTA
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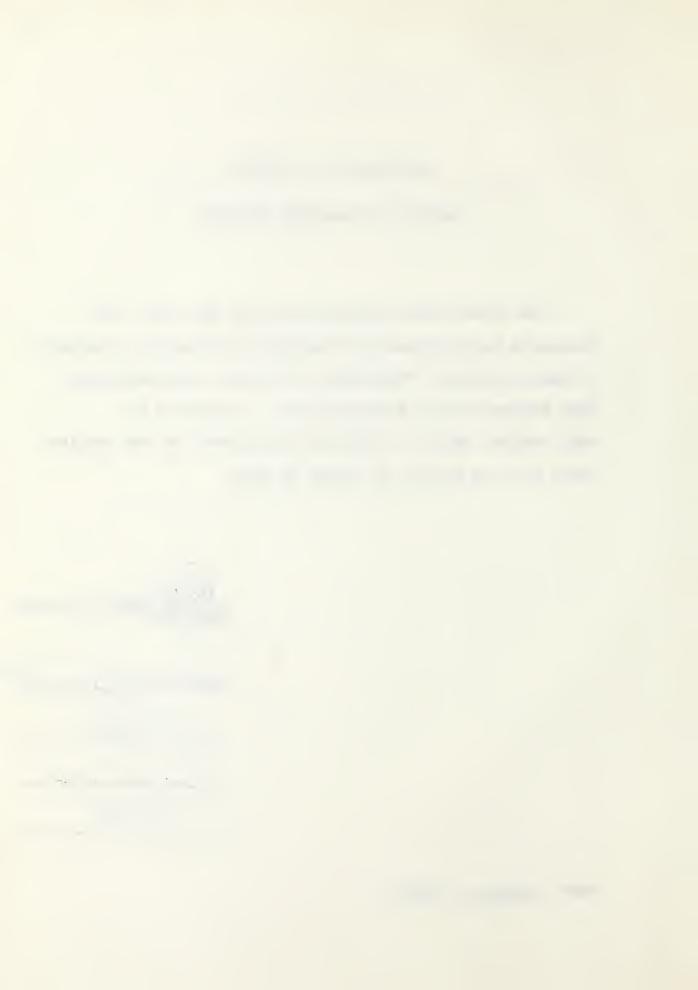


UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Effect of Alcohol and Amphetamine Upon Bender-Gestalt Reproductions", submitted by Neil Jeffrey Gamble in partial fulfillment of the requirements for the degree of Master of Arts.

Date May 1/ 1964



ABSTRACT

During the past half century much research has been concerned with the effects of alcohol on human behavior. In recent years there has also been research to determine the behavioral effects of the amphetamine compounds which act as general stimulants to the central nervous system. Few studies, however, have been done to determine the synergetic effects of alcohol and amphetamine on human personality and behavior.

In this study the Bender-Gestalt test was used to measure predicted personality changes under two drug conditions: alcohol and lactose, and alcohol and amphetamine. Measures of expansion on the Bender-Gestalt were taken as indications of a modification of inhibition or cortical control governing overt behavioral expression.

Specifically the hypotheses were:

- Subjects would produce significantly larger figure reproductions on the Bender-Gestalt under alcohol and amphetamine than under alcohol and placebo.
- 2) Subjects would produce significantly larger figure reproductions under alcohol and placebo than under non-drug conditions.
- 3) Subjects would produce significantly larger figure reproductions under alcohol and amphetamine than under non-drug conditions.



To test these hypotheses, twenty-five pharmacy and medical students were given the Bender-Gestalt over a four week period in the following sequence: pretest, two administrations under alcohol and lactose, two administrations under alcohol and amphetamine, posttest. Each subject received both drug combinations in a random order.

A further group of twenty-five medical and pharmacy students were given four administrations of the Bender-Gestalt over a four week period, under normal conditions. The results from this group were used to determine whether or not the measures used exhibited any significant practice effect.

The results confirmed all three hypotheses when an overall measure of expansion was considered. The conclusions were that alcohol does diminish inhibition or cortical control and that when amphetamine is used in combination with alcohol the effect is significantly intensified.



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CHAPTER I

INTRODUCTION

Statement of the problem.

Throughout history mankind has consumed alcohol in its many and varied forms. Until the past half-century, however, little was done to record scientifically the effects of ethyl alcohol on human physiological and psychological processes. Subsequent research indicated that although ethyl alcohol seemed to have a stimulating effect, it was in fact a depressant. The consequent euphoric state could be interpreted as being a result of the depressant alcohol lessening cortical "controls" through affecting those areas of the brain involved in inhibition, and allowing release of emotion and behavior.

In recent years a group of stimulant drugs, the amphetamines, have been widely studied. These drugs exert widespread stimulation and are generally classified on the basis of their predominant site of action within the central nervous system. Although many of the observable effects of amphetamine sulphate on man have been recorded, its physiological and psychological effects are still not completely understood. In general, the amphetamines appear to improve an individual's attentiveness, alertness, and awareness of his surroundings as well as inducing changes in mood similar to those caused by alcohol (Beecher, 1960a).

At the present time, few studies are available on the synergetic effect of alcohol and amphetamine in man. Research is



needed in this area because of the widespread use of amphetamine compounds as stimulants, especially amongst high school and college students. Beecher (1960a), for example, cited evidence that the amphetamines are being used by a growing number of young people to achieve "kicks" and that such a practice may contribute to antisocial behavior. Since the use of alcohol is also common amongst such groups there is a growing probability that alcohol and stimulants such as amphetamine, are being taken together, regardless of intention. Personality or behavior changes that might occur under these circumstances are, therefore, not only of scientific interest but also of practical importance.

The present study is primarily concerned with the effect of these drugs on inhibition. Sappenfield (1956), defined inhibition as "an automatic process that prevents the occurrence of overt actions" (p 216). It is in this sense that the term inhibition is used in this study.

The Bender-Gestalt test (B-G, hereafter) was the test selected to measure inhibition. This test is made up of nine geometrical figures which are presented to the subject to copy. Research, (Brengelmann, 1961), with this widely used clinical test, indicates that uninhibited and impulsive subjects are inclined to give expanded reproductions. Conversely behaviorally inhibited or overcontrolled subjects are known to give more



compressed (smaller and more restricted) figures.

Alcohol is thought to depress those areas of the brain controlling inhibition. Therefore, it is hypothesized that it should induce the subject to produce expanded figures on the B-G test. It may be ventured that alcohol produces euphoria by lessening inhibition and that amphetamine achieves the same result through energizing the system. Thus it would seem that the two achieve the same end by different means: in effect there is some complementarity. Thus the hypothesis offered is that the reactions with amphetamine in combination with alcohol should be similar to, but more intense than, the reactions under alcohol alone. This should cause subjects to show even greater expansion in their reproductions on the B-G.

In this present study, changes in size of figure reproduction on the B-G will be examined under two experimental drug conditions:

- 1) Standardized dosages of alcohol and lactose(as placebo).
- 2) Standardized dosages of alcohol and amphetamine.

Hypotheses

The hypotheses are that:

 Subjects will produce significantly larger figure reproductions on the B-G under alcohol and amphetamine than under alcohol and the placebo.



- 2) Subjects will produce significantly larger figure reproductions on the B-G under alcohol and the placebo than under non-drug conditions.
- 3) Subjects will produce significantly larger figure reproductions on the B-G under alcohol and amphetamine than under non-drug conditions.



CHAPTER II

DEFINITIONS AND RELATED LITERATURE

In this chapter will be discussed the nature of the test and the drugs used and relevant research will be reviewed. The first part of this chapter deals with ethyl alcohol in terms of its nature, classification and the metabolism involved in its assimilation. Thereupon, some attention will be given the research relating to its psychological effects. A similar outline will be followed in the second part which deals with amphetamine. The third section provides a brief summary of the research relevant to the synergetic effects of alcohol and amphetamine. The last part will allow discussion of the B-G and provide a rationale for its use in this study.

1. Ethyl Alcohol

Introduction

<u>Definition</u>. Pure ethyl alcohol is a colorless liquid which readily mixes with water and remains in solution. It is derived from the fermentation of glucose - a process which has as its final end products carbon dioxide and alcohol.

Classification. Alcohol is generally classified as a selective depressant. As early as 1883, the pharmacologist Schmiedeberg, challenged the commonly held belief that alcohol is a stimulant to the central nervous system. His theory was that the early observable effects following the ingestion of alcohol were due to a depressant effect on cerebral inhibition.



The later effects he equated to the spreading of depression to other functions. This theory has since earned general acceptance (de Ropp, 1960, p. 121).

Metabolism. After alcohol is ingested, it remains for a time in the stomach then passes to the duodenum. Some of the alcohol is absorbed from the stomach and the remainder is drawn rapidly through the walls of the small intestine. Alcohol is assimilated, unchanged, from the stomach and intestine in its passage into the blood stream, from which it is distributed evenly throughout the body. Absorption of the alcohol into the blood stream produces peak blood-alcohol levels in less than one hour. Those behavior changes especially characteristic of alcoholic intoxication, are produced when the alcohol in the blood stream reaches the brain.

Certain factors influence the rate of absorption. The higher the concentration of alcohol in the beverage (or dosage), the more rapid is the absorption. Because fat delays the emptying of the stomach, fatty food therein, slows absorption considerably.

<u>Dosages</u>. To achieve the same degree of concentration of alcohol in the blood stream from one individual to another, the alcohol to be ingested must be controlled according to body weight. It has been found empirically that 1.2 ml of 50% w/v



alcohol ingested per pound of body weight will produce approximately 100 mg per 100 ml of blood.

Psychological reactions

King (1957) outlined three main stages of intoxication and associated with each, characteristic psychological reactions. In the first stage the higher mental functions are impaired. In the second, motor and sensory functions are affected. Finally, the basic centres controlling the functions necessary for life are anesthetized.

Personality reactions

Most studies with alcohol have used a variety of measures for functions presumed to be affected by alcohol. Tests of general functions such as reaction time, co-ordination and muscle strength have been used. Still other tests of specific skills have been employed such as those measuring typewriting, target shooting, memory, continuous addition and numerical and verbal reasoning (see Trouton & Eysencks' review, 1960). However, according to Trouton and Eysenck (1960), studies of the effects of alcohol on personality have been largely ignored.

Miles (1932) set out a scale which relates certain subjective states and changes in behavior to varying percentages of alcohol in the blood. For example, without presenting the whole list, at the .05 level, that author described the imbibing



individual as "sitting on top of the world", an apparently free human being with very few inhibitions. Such a person operates on impulse, takes many personal and social liberties, often becomes belligerant, and expounds on his own virtues and exploits.

At this level, there is a relaxation of inhibitions resulting in the blunting of self-criticism. This reduction of inhibition under alcohol is considered to be due to the depressant effect of alcohol on those centres of the brain that customarily tend to inhibit spontaneous expression. As a consequence of these changes, the subjective experience is generally one of euphoria.

Phrased in terms of dynamic psychology, superego functions are diminished, resulting in a lessening of defencive activity and permitting more uninhibited expression. De Ropp (1957) describes the progress as follows:

"After alcohol has passed from the blood into the brain, it acts first on that area of the cerebral cortex which exerts a restraining action on our more native impulses, the censorious, restricting, critical entity to which Freudians have given the name of the Super-ego" (p. 121).

2. The Amphetamines

Introduction

<u>Definition</u>. Amphetamine is a volatile liquid in its natural state and belongs chemically to the group of phenylalkylamines,



isolated by Alles (1927). There are three clinical types of amphetamines:

- 1) methylamphetamine (Methedrine or Pervitin)
- 2) dextro-amphetamine sulphate (d-amphetamine or Dexedrine)
- 3) amphetamine sulphate (Benzedrine, Mecodrine and Isomyn)

Classification. Chemically and in their physiological effects, the amphetamines act as stimulants having an affect similar to adrenaline and noradrenaline. These are the hormones produced by the adrenal gland of vertebrates. Because these drugs stimulate a portion of the autonomic nervous system, Barger and Dale (1910) named their action sympathomimetic. Today they are classified clinically as stimulants.

Metabolism and dosage. Amphetamine sulphate is absorbed very rapidly from the intestines but metabolism proceeds slowly. Elimination of unaltered amphetamines apparently continues over a three to four day period. Beecher (1960a) cited an unpublished study in which he found the maximum effect of amphetamine achieved from two to three hours following ingestion. Dosage reactions however, are often highly individualistic.

Psychological reactions

As with alcohol, most studies on the effects of amphetamine on psychological functions have been concerned with factors of motor performance, memory, reasoning and learning. Again, as with alcohol, few studies have been concerned with the effects



of amphetamine on personality variables (Trouton & Eysenck, 1960).

In general, amphetamine produces an increased alertness, attentiveness, and a reduction of fatigue. Along with these changes a definite euphoria is experienced. Leake (1960), cited numerous studies showing the successful use of amphetamine in improving mood and producing a sense of confidence. It is used especially in the treatment of matinal, senescent, and pregnancy depression. Bradly and Bowen (1941) have demonstrated that the administration of amphetamine to lethargic withdrawn children produces more alertness, increased interest in the environment, and a greater appearance of happiness.

Beecher (1960a) in an extensive research project using college athletes, studied subjective evaluations of performance, physical state and mood under amphetamine. Employing a similar dosage (14-21 mg) to that employed in the present study, that author found certain subjective reactions with amphetamine. The subjects (hereafter, Ss) reported feelings of increased mental and physical activation; they also felt more friendly and found themselves less reserved in their social relationships. Finally, within the group there was a high frequency of reports of felt drunkenness, self satisfaction and impulsivity. These latter feelings would imply a reduction of



inhibition or control and are strikingly similar to feelings reported by subjects after drinking alcohol.

3. Alcohol and Amphetamine

Although amphetamine has been used to counteract depression and related symptoms which sometimes occur following heavy drinking (Alexander, 1953), there have been few studies carried out to determine the synergetic effects of alcohol and amphetamine in normal Ss. Newman and Newman (1956), carried out one such study to determine if caffeine and dexedrine would act as practical antagonists of the depressant effect of ethyl alcohol in man. The dosages of both alcohol and amphetamine employed were similar to those used in the present study. The authors found that on measures of balance, steadiness of hand, fusion frequency of flicker and on readings from an electroencephalogram, there were only small differences when performance under alcohol was compared to performance under both alcohol and amphetamine. Furthermore, the differences which were apparent exhibited no consistant direction. concluded that dexedrine in ordinary therapeutic doses was ineffective in a practical sense in combating the depressant effect of alcohol on the nervous system, in a normal group of The limitations of the study were a small normal human Ss. group (N = 6) and the lack of any statistical analysis of the data.



4. The Bender Visual Motor Gestalt Test (B-G)

Introduction

In the present study, expansive changes in the size of the figures reproduced on the B-G are taken as indices of lack of inhibition. Before reviewing the literature on the B-G, the relationship between personality and expressive movement deserves to be reviewed.

Expressive movement. Brengelmann (1960) defines expressive movement as follows:

"... expressive movement deals with the relation-ship of individual differences of involuntary motor behavior to personality and abnormality whether occurring during spontaneous or purposeful movements" (p. 62).

One of the assumptions underlying studies of expressive movement is that characteristic patterns of motor expression reflect personality structure and dynamics. Furthermore, changes in personality, whether transient or more permanent, are reflected by changes in patterns of motor expression (Anderson & Anderson, 1951, p. 288).

Size estimation, size of writing and drawing or size of figure reproduction as patterns of motor expression have been investigated. Allport and Vernon (1933), the pioneers in the field, found that Ss exhibited individual consistancy on general factors of expressive movement, including areal estimation. The main concern of these authors, however, was with the consistancy



of the factors rather than attempting to relate them to personality. Johnson (1937), was one of the first to study the relationship of expressive movement to personality. She found for example, that Ss were expansive on size estimation when euphoric and restrictive when depressed.

Bell (1948), Abt and Bellak (1950), Anderson and Anderson (1951) and Brengelmann (1960), provide excellent discussions of the subsequent developments in this field. The discussions by these authors relating the size factor in expressive movement to personality are best summarized by Brengelmann (1960).

"Writers usually agree that smallness and constriction are expressive of neurotic signs of depression, anxiety and compulsion; of feelings of inferiority and timidity; of rejection and deprivation; of introversion and emotional dependance. Large size and expansion are taken to indicate out-going, extraverted, aggressive and impulsive personalities. 'Over-controlled' persons for instance, draw small or constricted figures, even when they don't fit the description given above" (p. 75).

The Bender-Gestalt. The B-G is made up of nine geometric figures which the subject is required to copy. They were originally devised by Wertheimer (1923) in his studies on Gestalt laws of perception. Each figure was chosen because it embodied certain Gestalt principles. Gestalt theory is based on the assumption that perception is influenced by two factors:

1) the configuration of the stimuli presented, and, 2) the inherent biological organizing field forces in the brain. These



dynamic forces are continually at work modifying and integrating incoming stimuli. Because of these forces, the final perception may be more than a summation of the parts of the original stimulus.

Bender (1932) realized the potential of these designs as as task which could reveal pathological changes in the brain. She attempted to prove that in cases where maturation was incomplete, or with conditions such as brain damage, mental retardation, or certain psychotic conditions, the fundamental organizing tendencies in the brain would be disrupted. Furthermore, she anticipated these changes would be reflected in a more primitive performance on the B-G.

<u>Literature on the Bender-Gestalt</u>. Tolor and Schulberg (1963) critically reviewed the literature dealing with the B-G prior to 1961. They categorized the work as follows:

- 1) Reviews dealing with the rationale, history, administration and use (Bell, 1948; Woltmann, 1950; & Halpern, 1951).
- 2) Attempts to arrive at reliable systems of quantifying performance (Billingslea, 1948; Pascal & Suttell, 1950; Kitay, 1950; Peek & Quast, 1951 and Gobetz, 1953).
- 3) Attempts to differentiate various diagnostic syndromes using different scoring systems.
- 4) Attempts to relate certain aspects of B-G performance and personality dynamics.



This last mentioned area is particularly relevant to this thesis. A summary of the work done in relating personality and B-G performance follows, with particular emphasis on that work which has related size change to aspects of personality.

Bender-Gestalt and personality dynamics. In the foreword to the book by Tolor and Schulberg (1963), Bender commented on the lack of consistant, meaningful findings from studies attempting to relate B-G performance to personality. Her main criticisms of these studies were that personality constructs were not carefully enough defined and that not enough consideration was being given to the original Gestalt principles in the evaluation of B-G performance. She conceded, however, that research in this area could, if properly carried out, yield meaningful data.

Hutt (1945a) is one of the leading exponents in utilizing the B-G for revealing personality dynamics. His rationale for using the test in this manner is summed up in the following quotation:

"Because this test presents the subject with an apparently innocuous and neutral task, and because the final performance therefore, more intimately reveals the essential nature of his perceptual and adaptive behavior, it can be of significant or even crucial value in analyzing the psychodynamics of the personality in process" (p. 7).



Again:

"When a person is presented with nine geometrical figures in a relatively ambiguous situation and asked to make copies of them, he must proceed in a manner unique to his own past experience. His reactions reflect his 'style of life' and he will structure or deal with the task at hand in some way that approximates his tendencies to be himself" (Hutt & Briskin, 1960, p. 10)

Thus, if the individual is not impaired in terms of perceptual and/or motor ability, deviations that occur are thought to reflect factors other than Gestalt organizing-tendencies.

Hutt (1953) stated that interpretation should involve an attempt to ascertain the individual's characteristic adaptations and defences. In evaluating the B-G from this point of view, seven major categories are considered: organizational factors; determinants related to size; distortion of Gestalt; changes in form of Gestalt; movement determinants; miscellaneous factors and methods of work. In this present study only size changes are to be considered.

Size changes on the Bender-Gestalt. Hutt and Briskin, (1960), gives the following meaning for overall increases or decreases in the size of the figures reproduced on the B-G:

"... at the behavioral level over-all increase in size is related to outgoing, assertive behavior, while over-all decrease in size is related to withdrawing, passive behavior trends" (p. 58).



Again:

"Other findings indicate that persons with very strict superegos tend to reduce the size of their figures" (p. 55)

The following is a brief summary of the research directly relevant to Hutt's hypothesis, giving it some substantial support. Harriman and Harriman (1950) studied the relationship of B-G performance to reading readiness using nursery school and second grade children. They found that older children tended to exhibit less expansion in reproducing the figures. This would be the expectation, since younger children would have fewer controls and inhibitions.

Clawson (1959) used the B-G to determine if normal and malajusted children exhibited differences in performance, corresponding to differences in personality characteristics.

Some of her findings are as follows: 1) expansive B-G style is associated with acting out behavior in children,
2) compressed B-G style and withdrawn behavior are associated and 3) constricted B-G drawings are associated with constricted Rorschachs.

Using adult Ss, Gavales and Millon (1960) studied the relation of induced anxiety and the size of the design drawings. Their basic hypothesis was that reduction in size of drawing is more a function of the induced anxiety than of the ordinary differences in the anxiety levels of the Ss. Those authors



concluded that induced anxiety is associated with the drawing of smaller configurations. Furthermore, high basic anxiety level contributed to the reduction.

In a factor analytic investigation of the B-G, Guertin (1952), using one hundred patients at three mental hospitals, found that one of the factor loadings was constriction, which was thought to represent timidity and insecure feelings.

The experimental evidence strongly supports Hutt's hypothesis regarding the relationship between diminution changes in figure reproduction and inhibition.



CHAPTER III

METHOD

Subjects (Ss)

Two groups of volunteer Ss were employed: an experimental group and a normal group.

Experimental group (E). This group was made up originally of thirty-two medical and pharmacy students, involved in a larger experiment. Complete data, however, were obtained on only twenty-five of the original Ss. Inasmuch as alcohol was involved as an experimental condition, certain Ss could not participate: One S was a minor and was ineligible; one was a diabetic; one withdrew on personal grounds; and four others became ill during the course of the experiment.

Because the group was composed of volunteers, it was necessary to determine if they exhibited any unique characteristics. In terms of age, intelligence, and scores on personality tests, (Maudsley Personality Inventory and California Personality Inventory), the students in this group did not differ significantly from a group of 116 university students tested the previous year. ² In another previous study designed to test for

A large scale study recently conducted at the University of Alberta, to determine the effects of alcohol and amphetamine on various functions in man.

Personal communication with Professor Wilson, Department of Psychology, University of Alberta, 1964.



suggestibility in the use of amphetamine, only three of the twenty-five Ss proved to be reactors.

Normal group (N). This group was made up of twenty-five Ss drawn from the same classes as were the previous Ss in the experimental group. Significant differences were not found between the Ss in the E group and the N group on variables such as age, intelligence (Wonderlic) or scores on the Maudsley Personality Inventory (Table 1).

Table 1

As mentioned before, this present work is a part of a large scale experimental design. As such, it was impossible to properly select a control group. Thus, the normal group was added to the original design to suit immediate purposes. The normal group may be thought of then, as the best available measure of practice effect. It was not possible to assemble a perfect control group in this situation because of unavailability of students at the time the experimental group was being tested.

Experimental design: E group

A treatment by subject design was, used, in that every S received every treatment. In all, there were six B-G administrations spread over four sessions. Each of the four sessions libid.

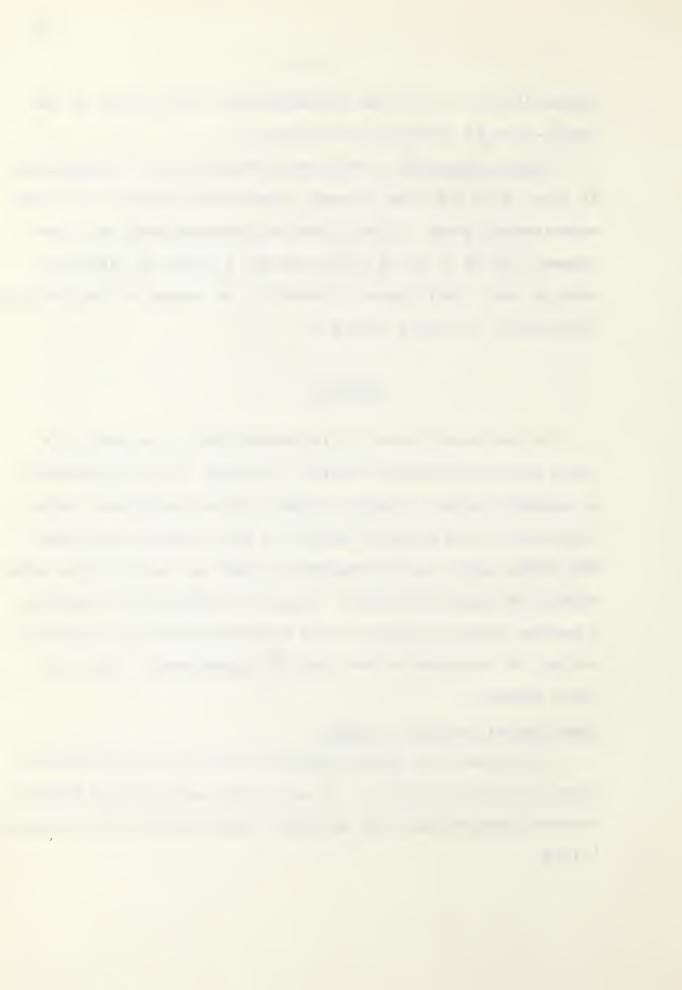


Table 1

COMPARISON OF E GROUP AND N GROUP

	E GROUP (N = 25)		N GROUP (N = 25)	
VARIABLE	Mean	S.D.	Mean	S.D.
Age	22.5	2	22.0	2.5
Wonderlic	31.2	5.2	31.4	3.5
Maudsley				
N	22.5	10.2	16.6	11.7
E	27.1	7.9	29.4	6.9

Note: \underline{t} tests were carried out on the difference between the means on each variable. None of the differences were significant.



was conducted at least one week apart and was always held in the afternoon from one to five. The test administrations included a pretest, two administrations under alcohol and lactose, two administrations under alcohol and amphetamine, and a posttest. The two administrations under alcohol and lactose and the two administrations under alcohol and amphetamine each took place, separately, during one experimental session. A "breathalyzer" reading was taken from each S prior to each administration of the B-G.

Every S received both of the combinations of drugs, receiving each in a randomized order. The drugs were given by code so that neither the person giving the tests nor the S knew which combination was being given. The administration of the drugs and the supervision of the lab were under the direction of the staff of the Department of Pharmacology, University of Alberta. The B-G was administered to each S individually by the author.

Pretest. At this time the Ss received a general orientation to the study. They also signed a statement indicating their willingness to participate as experimental Ss and abide by certain conditions, such as not leaving the lab during an experiment without authorization. For the remainder of the time, they familiarized themselves with some of the tasks they would have to perform in subsequent sessions. (See Appendix



A for description and order of other tests given Ss.) Each S also received his first administration of the B-G. Before leaving they were all told that they should eat a light, low-fat lunch prior to the experimental session II and III.

Alcohol and lactose. Each S was given sufficient rye whiskey (which contains flavouring agents in addition to ethanol) and ginger ale, orally, to provide 1.2 gm of ethyl alcohol per kgm of body weight. The final concentration in the beverage was 13.3% v/v. A capsule containing 315 mgm of lactose from a coded package was ingested together with the ethanol. The Ss were then instructed that they had thirty minutes to finish their drinks. After meeting this time requirement, the Ss began the sequence of experimental tasks. The first administration of the B-G was given 1½ hours after drinking was completed and again two hours later.

Alcohol and amphetamine. Each S received the same amount of alcohol in the same way as previously, except that this time a capsule containing 15 ml of amphetamine, in 300 mgm of lactose from a coded package was ingested together with ethanol. The Ss once more were given thirty minutes to drink the mixture. Following ingestion, the Ss carried out the same sequence of tasks as they had under alcohol and lactose. The B-G was given 1½ hours after the ingestion and again two hours later.



<u>Posttest</u>. The Ss took the B-G test in their final session.

Design N group

The twenty-five Ss in this group were given four administrations of the B-G over a three week period. In the first session they were given one administration; one week later, they were given two tests approximately two hours apart. The final administration was given the following week.

Measurement of blood-alcohol. In the present study the peak blood-alcohol level was reached in 12 hours (See Figure 3, p.39). A breathalyzer was used to determine blood-alcohol levels in the Ss. This instrument is designed to analyse the portion of ethyl alcohol excreted in the breath of people who have been drinking. Readings from the breathalyzer are obtained directly in terms of blood-alcohol concentration. The physiological fact that alcohol in alveolar air is in equilibrium with that in pulmonary blood makes this technical assessment possible. Begg, Hill and Nickolls (1964) for example, found a correlation of .98 between breathalyzer readings and bloodalcohol level determined by venous blood analysis. The advantages of the breathalyzer method of determining bloodalcohol levels are that it is simple to operate and it provides a means of obtaining continuous readings of blood-alcohol levels throughout an experiment.



Administration of the Bender-Gestalt

Each S was tested individually and received an 8½ x 11 sheet of paper and a sharp pencil. The instructions were given as follows: "I am going to show you some figures that I want you to copy." The examiner showed the nine B-G cards in the standard order, placing them one at a time at the top of the S's paper. If the S asked questions, the instructions were repeated. If he persisted in asking questions, the examiner replied: "Do it however you wish." The S was not allowed to move the cards or use any mechanical aids in reproducing the figures. After each test administration, the pencil was resharpened. If the S wished to use the reverse side of the paper, he was allowed to do so. No S asked for a second piece of paper.

Scoring the Bender-Gestalt

Since expansion of the reproduced figures was the important variable in this study, area was the central aspect of performance considered. The figures to be measured on the test protocols were first outlined so that area measures could be made. Some dimensions were not area measures but were included as possible indices of expansion. In addition, the total area of all the separate area measures was taken. In all, 24 separate measures were made on each test protocol.



For measuring the areas a Compensating Polar Planimeter was used which gives the area of any plane figure to 1/100 of a square inch. The actual measurements were made by a graduate geologist, trained in the use of the instrument and unfamiliar with the purpose of the experiment. Figure I indicates the measurements that were made on each of the nine B-G cards (card A and eight others).

Figure 1



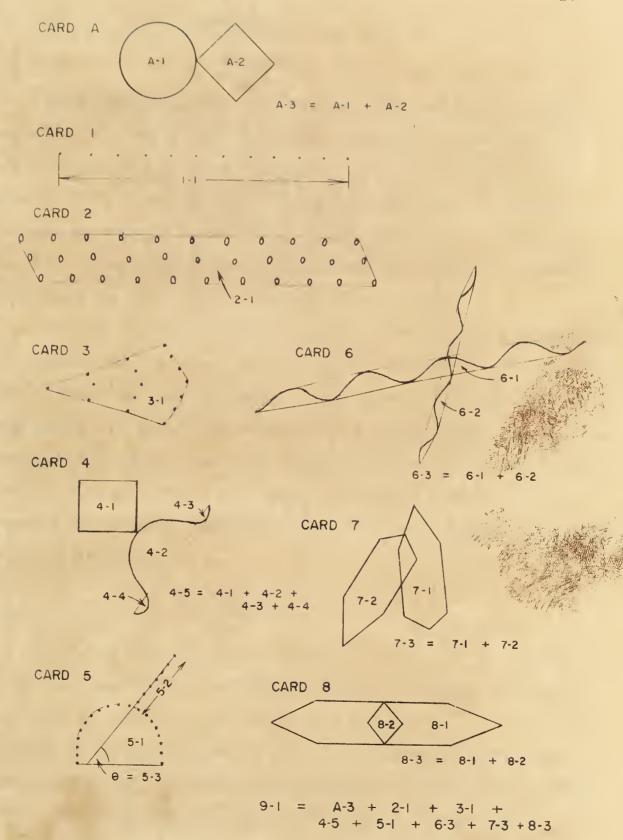


Fig. 1 Measures for Designs on Bender-Gestalt Cards (4/5ths original size)



CHAPTER IV

RESULTS

Experimental group. For each of the twenty-four measures employed, six means were obtained. Analyses of variance for a randomized block design (Edwards, 1960) was carried out to determine if there were any significant differences between any of the sets of means. When the <u>F</u> values were significant Duncan's (1955) new multiple range test was employed to determine which pairs of means differed significantly.

For all following tables certain abbreviations are employed. AL $_1$ represents the first administration of the B-G under alcohol and lactose, and AL $_2$ represents the second administration. AA $_1$ represents the first administration of the B-G under alcohol and amphetamine and AA $_2$ the second administration. The code figures used to designate the area measure are those used in Figure 1. Table 2 shows the \underline{F} values obtained when the analyses of variance were carried out.

Table 2

When the Duncan tests were made, fourteen of the measures exhibited significant differences (p. .05) between the pretest and each of the drug conditions (see Appendices C to H). The measure of overall expansion (9-1) showed highly significant differences (p. .001) between the pretest and each of the drug conditions (Appendix I). On the 5-2 measure there were



Table 2

SUMMARY OF ANALYSES OF VARIANCE

E GROUP

	MEASUREMENT	F*	SIGNIFICANCE LEVEL
CARD A	A-1 A-2 A-3	.58 1.98 1.02	N.S. N.S. N.S.
CARD 1	1-1	4.10	.01
CARD 2	2-1	10.09	.01
CARD 3	3-1	7.11	.01
CARD 4	4-1 4-2 4-3 4-4 4-5	3.01 3.94 2.36 6.15 4.95	.05 .01 .01 .01
CARD 5	5-1 5-2 5-3	11.81 10.06 1.23	.01 .01 N.S.
CARD 6	6-1 6-2 6-3	1.63 2.29 2.08	N.S. N.S. N.S.
CARD 7	7-1 7-2 7-3	4.50 3.57 4.85	.01 .01 .01
CARD 8	8-1 8-2 8-3	6.14 5.18 6.08	.01 .01 .01
TOTAL AREA	9–1	15.80	.01

^{*} see Appendices for means and complete analyses of variance tables.



significant differences (p \langle .05) on all but one of the pretest and drug condition comparisons (Appendix F). On the 1-1 measure there were significant differences (p \langle .05) on two of the pretest and drug condition comparisons (Appendix B). All the previously cited differences were in the predicted direction. On seven measures the \underline{F} values were not significant (Table 2). The pretest and posttest comparisons were not significant differences (p \langle .05) were found on seven measures within the drug condition comparisons. Table 3 summarizes the significant differences found and the direction of these differences.

Table 3

In order to present all of the experimental group findings in an integrated fashion for an easy general comprehension, an overall tabulation of the significant comparisons is presented in Table 4.

Table 4

Normal group. For each of the 24 measures employed, four means were obtained for this group. Again analyses of variance for a randomized block design (Edwards, 1960) were carried out.



Table 3

SUMMARY OF SIGNIFICANT DIFFERENCES FOUND

BETWEEN DRUG CONDITION COMPARISONS

E GROUP

MEASUREMENT	DRUG CONDITION COMPARISON	SIGNIFICANCE LEVEL
4-2	AA ₂ < AA ₁	.05
4-3	AL ₁ < AL ₂ AL ₁ < AA ₁ AL ₁ < AA ₂	.05 .05 .05
4-5	$\begin{array}{c} \text{AA}_2 < \text{AL}_1 \\ \text{AA}_2 < \text{AL}_2 \\ \text{AA}_2 < \text{AA}_1 \end{array}$.05 .05 .05
5-1	AL ₁ < AA ₂ AL ₁ < AA ₁	.05 .05
5-2	AL ₁ < AA ₂	.05
7-1	AA ₂ < AA ₁	.05
9-1	${\tt AL}_1<{\tt AA}_1$.05



SUMMARY OF SIGNIFICANT DIFFERENCES FOUND WITHIN PAIRED COMPARISONS:

E GROUP MEASURES

Table 4

	Significance Level for Measures																
Differences Tested	Card Card 2 Card 3				Card 4			Card 5		Card 7		Card 8			Card 9		
	- *	2-1	3-1	4-1	4-2	4-3	4-4	4-5	5-1	5-2*	7-1	7-2	7-3	8-1	8-2	8-3	9-1*
Pretest - Posttest	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	<i>N.S.</i>	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.Ş
Pretest - AA,	N.S.	·05	·05	·05	.05	·05	·05	·05	.05	·05	·05	·05	· <i>05</i>	·05	· <i>05</i>	·05	-001
Pretest - AA ₂	· <i>05</i>	· <i>05</i>	·05	.05	·0 5	· <i>05</i>	·0 5	·05	·05	·0 5	·05	· <i>05</i>	-05	·05	· <i>05</i>	·05	.001
Pretest - AL ₁	· <i>05</i>	·05	· <i>05</i>	-05	·05	·05	· <i>05</i>	·05	∙05	N.S.	· <i>05</i>	.05	·05	·0 5	·05	·05	001
Pretest - AL ₂	N.S.	-05	·0 5	·05	·05	·05	.05	·0 5	·05	·0 5	·05	·05	·05	·05	·0 5	· <i>05</i>	.001
AA ₁ - AA ₂	N.S.	N.S.	N.S.	N.S.	·05	N.S.	N.S.	·0 5	N.S.	<i>N.S.</i>	·0 5	N.S.	N.S.	N.S.	<i>N.S</i> .	N.S	N.S.
AL ₂ - AL ₂	N.S	N.S.	N .S.	N.S.	N.S.	·05	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
AL, - AA,	N.S.	N.S.	N.S.	N.S.	N.S.	∙05	N.S.	N.S.	.05	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	.05
AL ₂ - AA ₁	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S. ·	N.S.	N.S.	N .S.	N.S.	<i>N.S</i> .
AL ₁ - AA ₂	N.S.	<i>N.S</i> .	N.S.	N.S.	N.S.	·0 5	N.S.	·0 5	·05	·0 5	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.
AL ₂ - AA ₂	N.S.	N.S.	N.S.	N.S.	<i>N.S.</i>	N.S.	N.S.	·0 5	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

^{*} Linear measure (in inches)

^{* *} Total area measure



When the \underline{F} values were significant, Duncan's (1955) test was again carried out to determine which pairs of means differed significantly. Table 5 shows the F values obtained.

Table 5

Only five of the measures exhibited significant \underline{F} values. When Duncan's test was carried out on the means for each of the five measures, only six significantly different comparisons were found. Table 6 summarizes the significant differences found and the direction of these differences.

Table 6



Table 5
SUMMARY OF ANALYSES OF VARIANCE

N. GROUP

	MEASUREMENT	F*	SIGNIFICANCE LEVEL
CARD A	A-1 A-2 A-3	3.27 2.81 3.58	.05 .05 .05
CARD 1	1-1	.83	N.S.
CARD 2	2-1	1.94	N.S.
CARD 3	3-1	.94	N.S.
CARD 4	4-1 4-2 4-3 4-4 4-5	.17 .38 1.13 2.66 1.64	N.S. N.S. N.S. N.S.
CARD 5	5-1 5-2 5-3	.68 .73 .98	N.S. N.S. N.S.
CARD 6	6-1 6-2 6-3	4.71 1.56 4.27	.05 N.S. .05
CARD 7	7-1 7-2 7-3	.30 .69 .51	N.S. N.S. N.S.
CARD 8	8-1 8-2 8-3	.31 .34 .31	N.S. N.S. N.S.
TOTAL AREA	9-1	1.94	N.S.

^{*} see Appendix for means and complete analyses of variance tables.

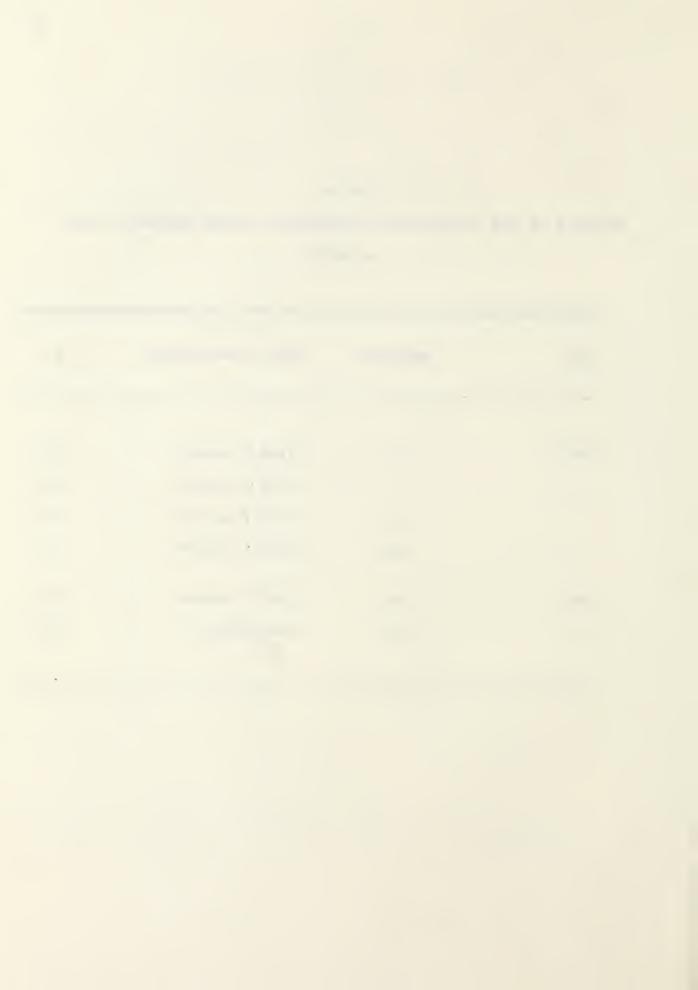


Table 6

SUMMARY OF THE SIGNIFICANT DIFFERENCES FOUND BETWEEN TRIALS

N.GROUP

CARD	MEASURE	TRIAL COMPARISON	p
CARD A	A-1	first < second	.05
		first (fourth	.05
	A-2	first < second	.05
	A-3	first < second	.05
CARD 6	6-1	first < second	.05
	6–3	fourth <pre>fourth</pre>	.05



CHAPTER V

DISCUSSION

Normal group

It was necessary to ascertain whether or not the measures employed exhibited a practice effect. From the foregoing (Table 6), it is apparent that the measures were not much influenced by practice and that therefore such effects are not likely to influence in any significant way the results in the experimental group.

Figure 2 shows the extent of the practice effect on the measure of overall expansion which, though it lacks significance, is interesting.

Figure 2

Experimental group

The first hypothesis stated that Ss would produce significantly larger figure reproductions on the B-G under alcohol and amphetamine than under alcohol and placebo. A summary of the significant differences and the direction of these differences found with six of the individual measures is given in Table 3. Additional individual measures failed to show any significant differences between paired comparisons of the drug conditions. A clear pattern did not emerge until a total area (9-1) was considered. When this was done the



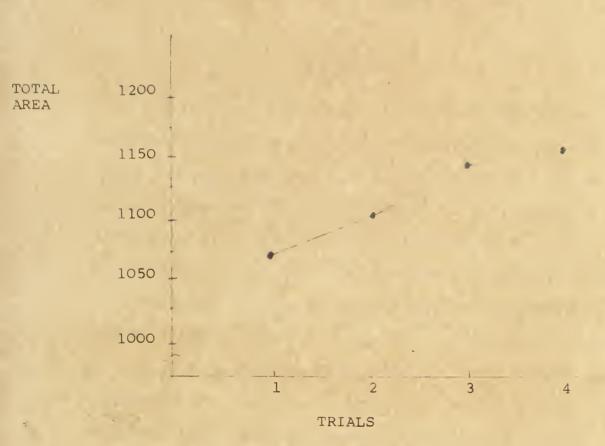


FIG. 2 - Total Area Measurement Over Four Trials

N Group



total area measure under AA_1 was found to be significantly larger (p \angle .05) than under AL_1 (Table 3).

The blood-alcohol levels found in the Ss $1\frac{1}{2}$ hours and $3\frac{1}{2}$ hours after the ingestion of both alcohol and lactose or alcohol and amphetamine are shown in Figure 3.

Figure 3

Significant differences in blood-alcohol levels were not found between AL_1 and AA_1 or between AL_2 and AA_2 . This would indicate that the significant increase found in overall area between AL_1 and AA_1 cannot be attributed to a difference in alcohol levels. Since $AA_1 > AL_1$ it can be postulated further that the increase in expansion under AA_1 is a result of the potentiating effect of amphetamine and alcohol rather than any change in blood-alcohol level.

Significant differences were not found between AL_2 and AA_2 when total area was considered. It can be seen from Figure 4 that although expansion between AA_1 and AA_2 decreased there was an increase in expansion between AL_1 and AL_2

Figure 4

Although neither of these changes was statistically significant, a smaller and still not significant difference



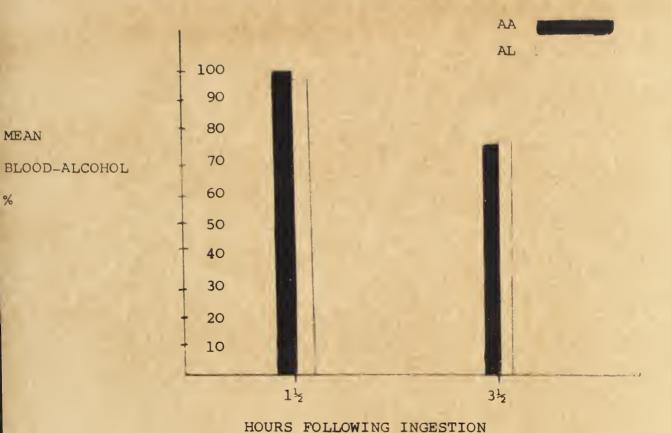
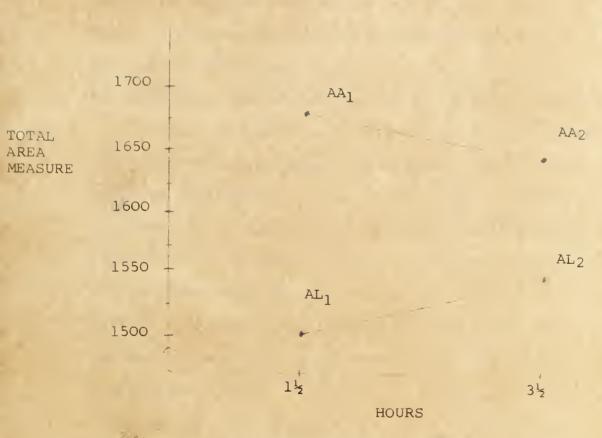


FIG. 3 - Mean Blood-Alcohol % During
Bender-Gestalt Administration





TIME OF BENDER GESTALT ADMINISTRATION

FIG. 4 - Total Area Measure at $1\frac{1}{2}$ and $3\frac{1}{2}$ Hours Following Ingestion of Drugs



between AL2 and AA2 resulted and is notable. This may be that although the amphetamine with the alcohol produces a greater expansion initially, that its presence accounts for a diminution within that condition; as contrasted with the condition wherein amphetamine is lacking: there is, in fact, an expansive trend under alcohol and lactose. Since measures of amphetamine levels in the Ss could not be taken, the foregoing must be considered as somewhat speculative.

In the introduction to this study it was suggested that a potentiating effect would occur under the drug combination because both drugs given singly produced euphoria. Furthermore, it was speculated that each drug produced the euphoria in a different manner. A further speculative thought can be added regarding the processes involved in the combined action of the two drugs. Alcohol may depress the inhibitory centres of the brain which check impulsive behavior through biochemical action. Amphetamine, producing an electrical discharge up into the cortex, may not stimulate the inhibitory centres enough to over-ride the effects of the alcohol. general cortical arousal produced, however, would intensify affective and overt behavioral expression. Furthermore, over a period of time, the amphetamine may start to over-ride the effects of the alcohol on the inhibitory centres producing a diminution of the original effect. If this were so, it would



explain the trend seen in Figure 4. It must be appreciated by the reader that at present, there is little direct evidence to fully support these speculations.

Having stated all this, it can be said that Ss produce significantly larger reproductions of the B-G figures under alcohol and amphetamine than under alcohol and lactose. However, this appears to be more especially true if the measure of expansion is taken at the $l\frac{1}{2}$ hour point following ingestion.

The second hypothesis stated that Ss would produce significantly larger figure reproductions on the B-G under alcohol and placebo than under a non-drug condition. With 14 of the individual measures there were significant differences (p < .05) between the pretest and each of the AL₁ and AL₂ conditions (Appendices C to H). All differences were in the predicted direction. Two linear measures 1-1 and 5-2 failed to show significant differences on at least one of the comparisons (Appendices B and F). Measure 5-3, which was an angle measure, failed to exhibit any significant differences (Appendix F). A possible explanation of these latter findings is that non-area measures may be less reliable indicators of expansion than are those involving area. Six area measures (6-1, 6-2, 6-3, A-1, A-2, and A-3) were non-significant with every comparison (Table 2). Since these same measures, with the exception of 6-2, were the only measures that showed



significant variation between administrations with the normal group, (Table 6), it may be that they are more determined by non-experimental variables than the other area measure employed. A possible explanation for these findings with A-1, A-2 and A-3 is that Figure A by intention is an easier one to reproduce and Ss generally commence the task with better control. It is possible that the Ss in the E group, when tested under drug conditions, were attempting to counteract expansive tendencies and maintain their previous control on this first figure more than with subsequent figures. In comparison, the Ss in the N group apparently were exerting stronger control in reproducing figure A on the first trial while they were unfamiliar with the task, than on subsequent trials. A possible reason for the variability of 6-1, 6-2, and 6-3 may be the way in which the figures can exhibit linear or vertical expansion without the area varying, providing there is not an increase in both.

When total area measure (9-1) was considered, all comparisons between pretest and AL conditions were significant at the .001 level and were in the predicted direction (Appendix I). Thus, this measure, which was the best measure of overall expansion, fully supports the second hypothesis.

The third hypothesis stated that the Ss would produce significantly larger figure reproductions under alcohol and



amphetamine than under non-drug conditions. When the two conditions, alcohol and amphetamine and alcohol and lactose and their pretest, were compared for individual measures, the findings were almost identical. The only difference found in this respect was with 5-2. Here the mean areas for AA₁ and AA₂ were each significantly larger than the pretest mean area, whereas with alcohol and lactose only one of the AL comparisons had been significant (Appendix F).

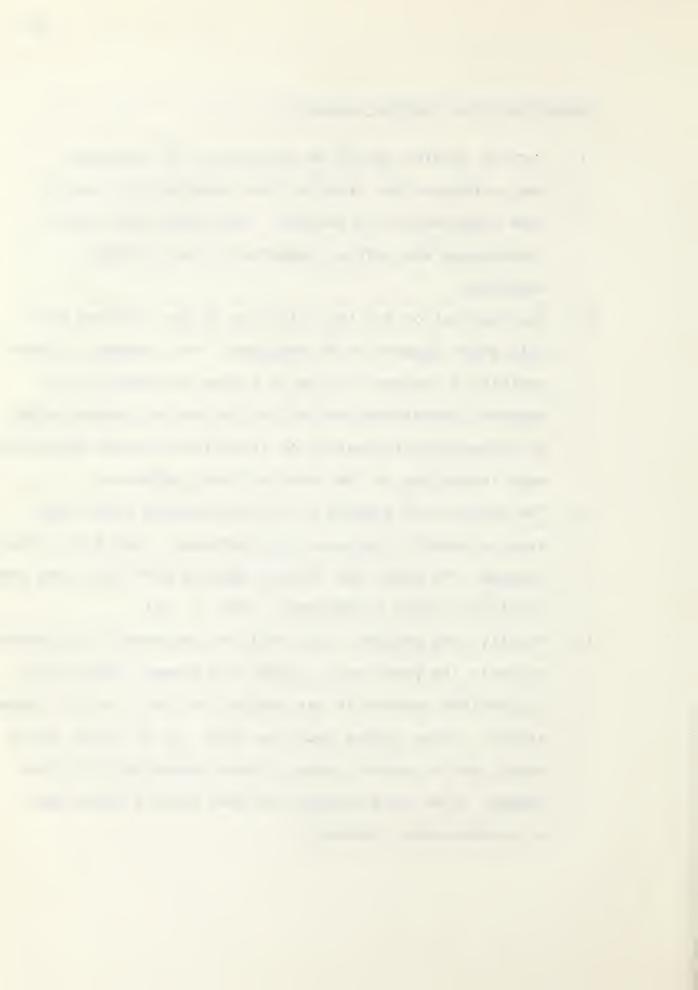
When the total area measure (9-1) was considered, the differences were highly significant at the .001 level for all comparisons and were in the predicted direction, confirming the third hypothesis.

From the foregoing then, it can be stated that all three hypotheses were confirmed when a measure of overall expansion was considered. The present study thus provided further experimental evidence for the relationship that Hutt and Briskin (1960) postulated between diminution in size of figure reproduction on the B-G and inhibition. Furthermore, the study has shown that this aspect of B-G performance is sensitive to temporary changes in inhibition produced by drugs. Lastly, the results indicated that compared to alcohol, amphetamine and alcohol produced a decrease in inhibition as measured by size of figure reproduction on the B-G.



Suggestions for further research

- 1) Further studies should be carried out to determine what effects other drugs or drug combinations have on size reproduction on the B-G. One such study should investigate what effect amphetamine has on these measures.
- The implication for the clinician of the findings from this study deserve to be explored. For example, clients exhibiting changes in size of figure reproduction on repeated administrations of the B-G during therapy might be reflecting fluctuation of inhibition, thereby providing some indication of the state of their defences.
- 3) The relation of aspects of B-G performance other than size to specific personality hypotheses, should be investigated. To date, few studies dealing with this area are available (Tolor & Schulberg, 1963, p. 46).
- 4) Finally, the present study could be repeated using better controls (As previously stated, the present design had limitations because it was adapted to fit in with a larger study). Three groups could be used: an AA group, an AL group, and a control group. These groups would be pretested, given drug dosages and then given a final test on an equivalent schedule.



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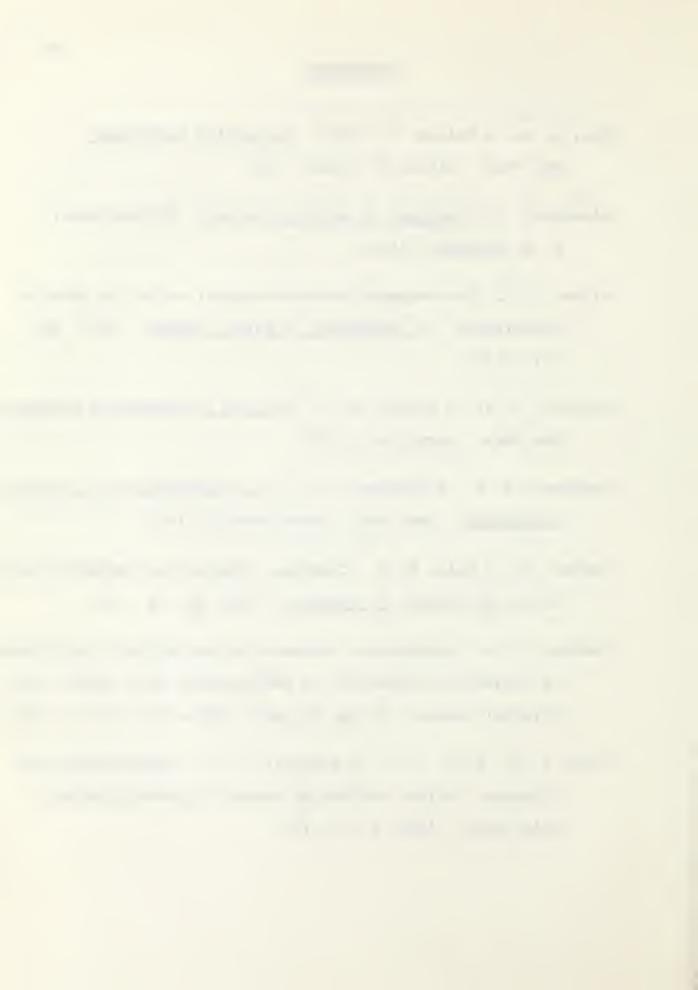
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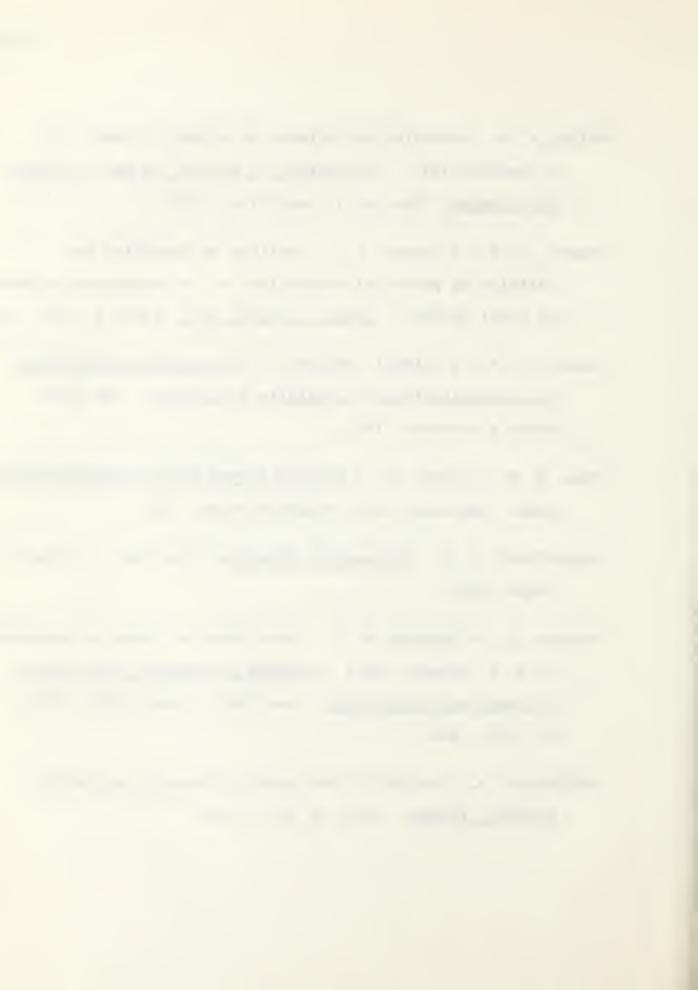
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APPENDICES



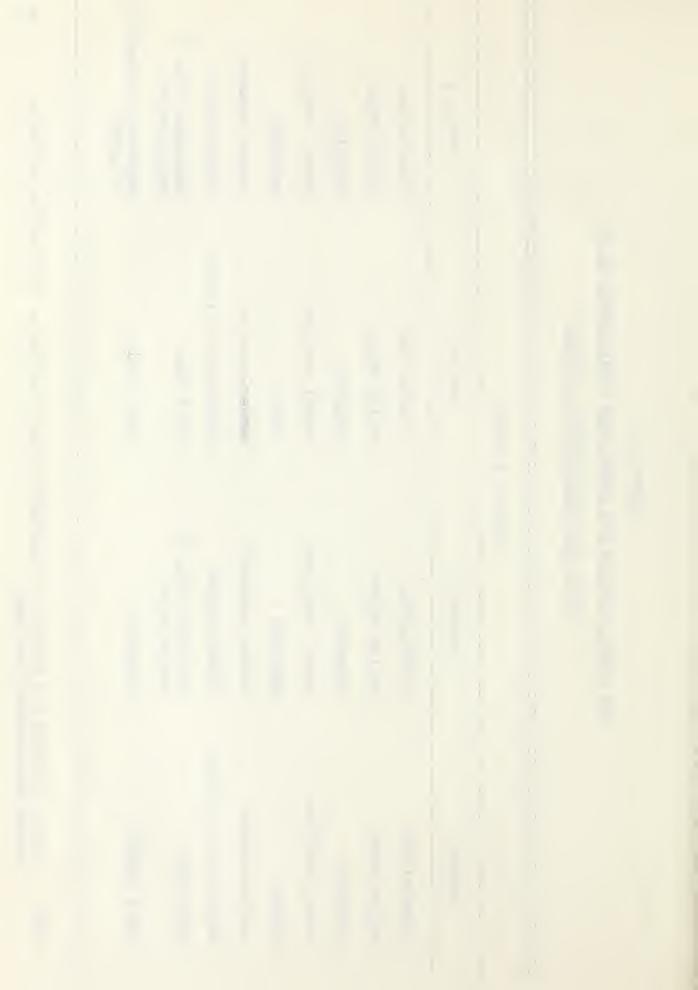
THE ORDER IN WHICH THE TESTS WERE ADMINISTERED TO SS

DURING EACH EXPERIMENTAL SESSION

Series of Tests*

Series 4	Breathalyzer	Police Tests	Skipping	Mental Addition	Decoding	Purdue Pegboard	Pursuit Rotor	Minnesota Rate of Manipulation	Maudsley Personality Inventory
Series 3	Breathalyzer	Police Tests	Skipping	Mental Addition	Coding	Bender-Gestalt	Muller-Lyer Illusion	Digit Span	Trail Making
Series 2	Breathalyzer	Police Tests	Skipping	Mental Addition	Decoding	Purdue Pegboard	Pursuit Rotor	Minnesota Rate of Manipulation	Wonderlic
Series l	Breathalyzer	Police Tests	Skipping	Mental Addition	Coding	Bender-Gestalt	Muller-Lyer Illusion	Digit Span	Trail Making

Each series of tests The Ss finished one series and immediately began the next. lasted approximately one hour. NOTE



Appendix B

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURE

CARD 1 - E GROUP

Difference Technol	Cimificance Issuel for Monayan				
Differences Tested	Significance Level for Measure				
	1-1 *				
Pretest - Posttest	N.S.				
Pretest - AA ₁	N.S.				
Pretest - AA ₂	.05				
Pretest - AL ₁	.05				
Pretest - AL2	N.S.				
$AA_1 - AA_2$	N.S.				
AL ₁ - AL ₂	N.S.				
AL ₁ - AA ₁	N.S.				
AL ₂ - AA ₁	N.S.				
AL ₁ - AA ₂	N.S.				
AL ₂ - AA ₂	N.S.				

^{* 1-1} is measured in inches



Appendix C

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURE

CARD 2 - E GROUP

Differences Tested	Significance Level for Measure			
	2–1			
Pretest - Posttest	N.S.			
Pretest - AA ₁	.05			
Pretest - AA ₂	.05			
Pretest - AL ₁	.05			
Pretest - AL2	.05			
AA ₁ - AA ₂	N.S.			
AL ₁ - AL ₂	N.S.			
AL ₁ - AA ₁	N.S.			
AL_2 - AA_1	N.S.			
$AL_1 - AA_2$	N.S.			
AL ₂ - AA ₂	N.S.			



Appendix D DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURE

CARD 3 - E GROUP

Differences Tested	Significance Level for Measure			
	3-1			
Pretest - Posttest	N.S.			
Pretest - AA ₁	.05			
Pretest - AA ₂	.05			
Pretest - AL ₁	.05			
Pretest - AL2	.05			
AA ₁ - AA ₂	N.S.			
AL ₁ - AL ₂	N.S.			
AL ₁ - AA ₁	N.S.			
AL ₂ - AA ₁	N.S.			
AL ₁ - AA ₂	N.S.			
AL ₂ - AA ₂	N.S.			



Appendix E

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURES

CARD 4 - E GROUP

Differences Tested Significance Level for Measures					
	_			4-4	
Pretest - Posttest	N.S.	N.S.	N.S.	N.S.	N.S.
Pretest - AA ₁	.05	.05	.05	.05	.05
Pretest - AA ₂	.05	.05	.05	.05	.05
Pretest - AL ₁	.05	.05	.05	.05	.05
Pretest - AL2	.05	.05	.05	.05	.05
AA ₁ - AA ₂	N.S.	.05	N.S.	N.S.	.05
AL ₁ - AL ₂	N.S.	N.S.	.05	N.S.	N.S.
AL ₁ - AA ₁	N.S.	N.S.	.05	N.S.	N.S.
AL ₂ - AA ₁	N.S.	N.S.	N.S.	N.S.	N.S.
AL ₁ - AA ₂	N.S.	N.S.	.05	N.S.	.05
AL ₂ - AA ₂	N.S.	N.S.	N.S.	N.S.	.05



AppendixF DUNCAN'S NEW MULTIPLE RANGE TEST: PAIRED COMPARISONS FOR MEASURES

CARD 5 - E GROUP

Differences Tested	Signifi	Significance Level for Measures			
	5-1	5-2*	5-3**		
Pretest - Posttest	N.S.	N.S.	N.S.		
Pretest - AA ₁	.05	.05	N.S.		
Pretest - AA ₂	.05	.05	N.S.		
Pretest - AL ₁	.05	N.S.	N.S.		
Pretest - AL2	.05	.05	N.S.		
$AA_1 - AA_2$	N.S.	N.S.	N.S.		
AL ₁ - AL ₂	N.S.	N.S.	N.S.		
AL ₁ - AA ₁	.05	N.S.	N.S.		
AL ₂ - AA ₁	N.S.	N.S.	N.S.		
AL ₁ - AA ₂	.05	.05	N.S.		
AL ₂ - AA ₂	N.S.	N.S.	N.S.		

^{* 5-2} is measured in inches
** 5-3 is measured in degrees



Appendix G

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURES

CARD 7 - E GROUP

Differences Tested	Signific	Significance Level for Measures			
	7-1	7–2	7–3		
Pretest - Posttest	N.S.	N.S.	N.S.		
Pretest - AA ₁	.05	.05	.05		
Pretest - AA ₂	.05	.05	.05		
Pretest - AL ₁	•05	.05	.05		
Pretest - AL2	.05	.05	.05		
AA ₁ - AA ₂	.05	N.S.	N.S.		
AL ₁ - AL ₂	N.S.	N.S.	N.S.		
AL ₁ - AA ₁	N.S.	N.S.	N.S.		
AL ₂ - AA ₁	N.S.	N.S.	N.S.		
AL ₁ - AA ₂	N.S.	N.S.	N.S.		
AL ₂ - AA ₂	N.S.	N.S.	N.S.		



Appendix H

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURES

CARD 8 - E GROUP

Differences Tested	Signific	Significance Level for Measures			
	8–1	8-2	8-3		
Pretest - Posttest	N.S.	N.S.	N.S.		
Pretest - AA ₁	.05	.05	.05		
Pretest - AA ₂	.05	.05	.05		
Pretest - AL ₁	.05	.05	.05		
Pretest - AL2	.05	.05	.05		
AA ₁ - AA ₂	N.S.	N.S.	N.S.		
AL ₁ - AL ₂	N.S.	N.S.	N.S.		
AL ₁ - AA ₁	N.S.	N.S.	N.S.		
AL ₂ - AA ₁	N.S.	N.S.	N.S.		
AL ₁ - AA ₂	N.S.	N.S.	N.S.		
AL ₂ - AA ₂	N.S.	N.S.	N.S.		



Appendix I

DUNCAN'S NEW MULTIPLE RANGE TEST:

PAIRED COMPARISONS FOR MEASURE

CARD 9 - E GROUP

Differences Tested	Significance Level for Measure
	9–1
Pretest - Posttest	N.S.
Pretest - AA ₁	.001
Pretest - AA ₂	.001
Pretest - AL ₁	.001
Pretest - AL2	.001
AA ₁ - AA ₂	N.S.
AL ₁ - AL ₂	N.S.
AL ₁ - AA ₁	.05
AL ₂ - AA ₁	N.S.
AL ₁ - AA ₂	N.S.
AL ₂ - AA ₂	N.S.



Appendix J

MEAN AREAS FOR MEASURES

CARD A - E GROUP

Candibian	ida a sa C	116	
Condition	A-1	or Measu A-2	re A-3
Pretest	. 54	.37	.92
AL_1	.63	.43	1.05
AL ₂	.57	.42	.98
AA 1	. 60	.46	1.06
AA ₂	.55	.38	.93
Posttest	.58	.34	.92



Appendix K

MEAN LENGTHS FOR MEASURE

CARD 1 - E GROUP

Condition	Mean for Measure
	1-1 *
Pretest	4.43
AL ₁	4.85
AL ₂	4.67
AAl	4.79
AA ₂	4.82
Posttest	4.21

^{* 1-1} is measured in inches



Appendix L MEAN AREAS FOR MEASURE CARD 2 - E GROUP

Condition	Mean for Measure		
V	2-1		
Pretest	2.24		
AL ₁	2.74		
AL ₂	2.80		
AA_1	3.06		
AA ₂	3.24		
Posttest	2.08		



Appendix M MEAN AREAS FOR MEASURE

CARD 3 _ E GROUP

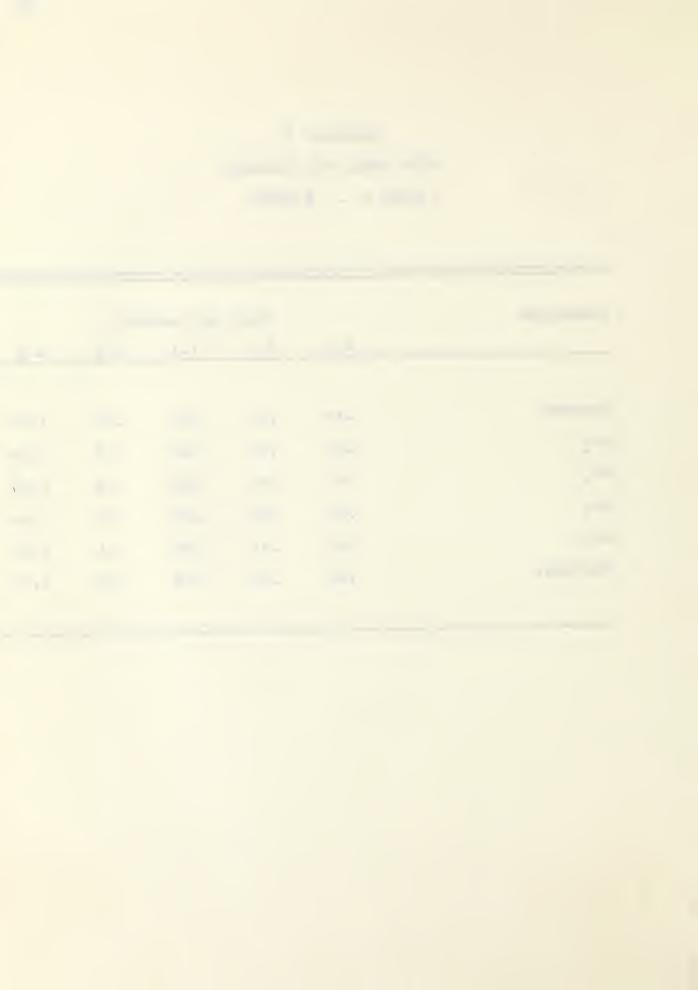
Condition	Mean for Measure
	3–1
Pretest	1.07
AL ₁	1.34
AL ₂	1.41
AAl	1.46
AA ₂	1.60
Posttest	1.02



Appendix N
MEAN AREAS FOR MEASURES

CARD 4 - E GROUP

Condition		Mean for Measure			
***	4-1	4-2	4-3	4-3	4-5
Pretest	.48	.43	.05	.07	1.03
AL ₁	.59	.59	.08	.13	1.39
AL ₂	.62	.61	.08	.14	1.47
AA ₁	.62	.66	.08	.12	1.48
AA ₂	.65	.51	.08	.11	1.35
Posttest	.56	.46	.05	.05	1.12



Appendix 0 MEAN SCORES FOR MEASURES

CARD 5 - E GROUP

Condition	Mean for Measure			
	5-1	5-2*	5-3**	
Pretest	1.08	1.07	.48	
AL ₁	1.36	1.18	. 47	
AL ₂	1.51	1.30	.46	
AA ₁	1.74	1.30	. 45	
AA ₂	1.73	1.36	.43	
Posttest	.96	.99	. 45	

^{* 5-2} is measured in inches
** 5-3 is measured in degrees



Appendix P

MEAN AREAS FOR MEASURES

CARD 6 - E GROUP

Condition	Mean for Measure			
	6-1	6–2	6–3	
Pretest	1.40	.76	2.16	
AL ₁	1.72	1.00	2.72	
AL ₂	1.68	1.04	2.68	
AA1	1.85	.98	2.83	
AA ₂	1.81	1.04	2.85	
Posttest	1.58	.78	2.36	



Appendix Q

MEAN AREAS FOR MEASURES

CARD 7 - E GROUP

Condition	Mean fo	r Measure	
	7-1	7–2	7–3
Pretest	.99	.78	1.77
AL ₁	1.26	.99	2.24
AL ₂	1.33	1.07	2.40
$^{\mathrm{AA}}$ 1	1.50	1.14	2.63
AA ₂	1.24	1.02	2.26
Posttest	1.02	.92	1.94



Appendix R

MEAN AREAS FOR MEASURES

CARD 8 - E GROUP

Condition	Mean i	Mean for Measure			
	8–1	8-2	8-3		
Pretest	1.47	.14	1.61		
AL ₁	1.98	.22	2.20		
AL2	1.94	.22	2.17		
AA 1	2.35	.24	2.59		
AA ₂	2.24	.22	2.46		
Posttest	1.57	.13	1.69		



Appendix S

MEAN AREAS FOR MEASURE

9-1 – E GROUP

Condition	Mean for Measure		
	9–1		
Pretest	11.89		
AL ₁	15.05		
AL ₂	15.46		
AA ₁	16.85		
AA ₂	16.47		
Posttest	12.09		

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Appendix T
ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD A _ E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure A-1				
Treatments	.12	5	.025	.589
Blocks	6.35	24	.264	
Residual	5.10	120	.042	
TOTAL	11.57	149		
Measure A-2				
Treatments	.22	5	.044	1.98
Blocks	2.60	24	.108	
Residual	2.70	120	.022	
TOTAL	5.52	149		
Measure A-3				
Treatments	53	5	.106	1.02
Blocks	16.25	24	.677	
Residual	12.58	120	.104	
TOTAL	29.36	149		



Appendix U

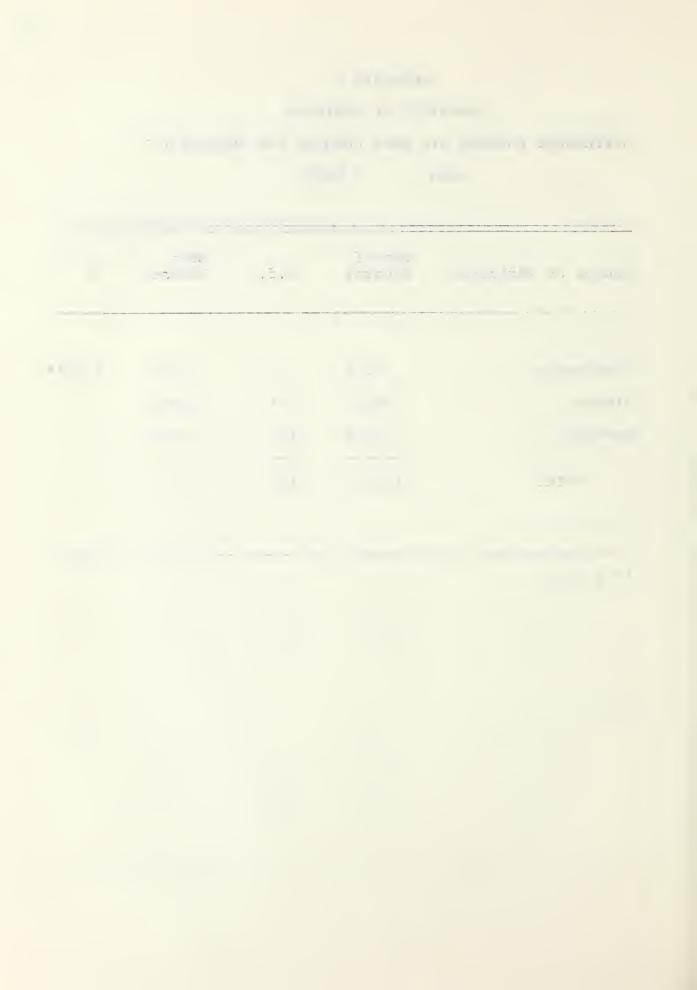
ANALYSIS OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN LENGTHS FOR MEASURE 1-1

CARD 1 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
	*			
Treatments	8.26	5	2.857	4.10**
Blocks	68.57	24	1.653	
Residual	48.33	120	.402	
TOTAL	125.16	149		

^{**} p<.01



Appendix V

ANALYSIS OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURE 2-1

CARD 2 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Treatments	25.65	5	5.131	10.09**
Blocks	63.09	24	2.629	
Residual	61.04	120	.508	
TOTAL	149.78	149		

^{**} p < .01



Appendix W

ANALYSIS OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURE 3-1

CARD 3 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Treatments	6.55	5	1.311	7.11**
Blocks	19.03	24	.793	
Residual	22.13	120	.184	
				
TOTAL	47.71	149		

^{**} p < .01



Appendix X

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 4 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
	•		•	
Measure 4-1				
Treatments	.44	5	. 089	3.01*
Blocks	4.62	24	.192	3.01
Residual	3.56	120	.029	
TOTAL	8.62	149		
Measure 4-2	٠			
Treatments	1.04	5	2.08	3.94**
Blocks	5.54	24	2.31	
Residual	6.34	120	.052	
TOTAL	12.92	149		
Measure 4-3				
Treatments	• 03	5	.006	2.36*
Blocks	.23	24	.009	
Residual	•31	120	.002	
TOTAL	.57	149		

^{*} p < .05 ** p < .01



Appendix Y

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 4 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
	* * * ** · · · · · · · · · · · · · · ·			
Measure 4-4				
Treatments	.16	5	.030	6.15**
Blocks	.72	24	.033	
Residual	.65	120	.005	
TOTAL	1.53	149		
Measure 4-5				
Treatments	4.32	5	.864	4.95**
Blocks	22.13	24	.922	
Residual	20.95	120	.174	
TOTAL	47.40	.49		

^{**} p< .01

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Appendix Z
ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN SCORES FOR MEASURES

CARD 5 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 5-1				
Treatments	13.44	5	2.688	11.81**
Blocks	44.58	24	1.857	
Residual	27.31	120	.227	
TOTAL	85.33	149		
Measure 5-2				
Treatments	2.64	5	•528	10.06**
Blocks	6.96	24	.290	
Residual	6.30	120	.052	
TOTAL	15.90	149		
Measure 5-3			<u> </u>	
Treatments	.04	5	.007	1.23
Blocks	•58	24	.024	
Residual	.72	120	.006	
TOTAL	1.33	149		

^{**} p < .01

Note: 5-2 is measured in inches

5-3 is measured in degrees

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Appendix A A
ANALYSES OF VARIANCE

CARD 6 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 6-1				
Treatments	3.34	5	.669	1.63
Blocks	78.10	24	3.254	
Residual	49.17	120	.409	
TOTAL	130.61	149		
Measure 6-2				
Treatments	2.11	5	.422	2.29
Blocks	19.89	24	.828	
Residual	22.19	120	.184	
TOTAL	44.20	149		
Measure6-3				
Treatments	9.67	5	1.935	2.08
Blocks	147.35	24	6.139	
Residual	111.73	120	.931	
TOTAL	268.75	149		



Appendix B B
ANALYSES OF VARIANCE

CARD 7 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 7-1		algani i Marianga anna anna anna anna anna anna		***
Treatments	4.57	5	.914	4.50**
Blocks	17.74	24	.739	
Residual	24.37	120	.203	
TOTAL	46.68	149		
Measure 7-2				
Treatments	1.96	5	.392	3.57**
Blocks	10.37	24	.432	
Residual	13.19	120	.109	
TOTAL	25.54	149		
Measure 7-3				
Treatments	12.12	5	2.424	4.86**
Blocks	50.16	24	2.090	
Residual	59.83	120	.498	
TOTAL	122.11	149		

^{**} p<.01



Appendix C C
ANALYSES OF VARIANCE

CARD 8 _ E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 8-1				
Treatments	15.42	5	3.085	6.14**
Blocks	75.55	24	3.148	
Residual	60.27	120	.502	
TOTAL	151.24	149		
Measure 8-2				
Treatments	.28	5	.056	5.18**
Blocks	1.06	24	.044	
Residual	1.30	120	.010	
TOTAL	2.64	149		
Measure 8-3				
Treatments	19.58	5	3.916	6.08**
Blocks	92.32	24	3.846	
Residual	77.29	120	.644	
TOTAL	189.19	149		

^{**} p <.01



Appendix D D

ANALYSIS OF VARIANCE

9-1 - E GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Treatments	578.50	5	115.700	15.80**
Blocks	1290.45	24	53.768	
Residual	878.96	120	7.324	
TOTAL	2747.91	149		
20112	2717.51	2.10		

^{**} p < .01

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Appendix E E

MEAN AREAS FOR MEASURES

CARD A - N GROUP

Administration	Mean for	r Measur	е
	A-1	A-2	A-3
First	.46	.30	.76
Second	. 60	.38	.98
Third	.55	.35	.88
Fourth	.60	.36	. 95



Appendix F F MEAN LENGTHS FOR MEASURE CARD 1 - N GROUP

Administration	Mean for Measure
	1-1*
First	4.38
Second	4.48
Third	4.50
Fourth	4.66

^{* 1-1} is measured in inches



Appendix G G MEAN AREAS FOR MEASURE CARD 2 - N GROUP

Administration	Mean for Measure
	2–1
First	2.10
Second	2.25
Third	2.38
Fourth	2.32



Appendix H H MEAN AREAS FOR MEASURE CARD 3 - N GROUP

Administration	Mean for Measure
	3-1
First	.94
Second	1.03
Third	•97
Fourth	1.05



Appendix I I

MEAN AREAS FOR MEASURES

CARD 4 - N GROUP

Administration	Ме	ean for N	Measure		
	4-1	4-2	4-3	4-4	4-5
First	.45	.46	.04	.05	1.01
Second	. 50	. 45	.04	.05	1.04
Third	• 50	.44	.04	.05	1.04
Fourth	•56	.48	.05	.07	1.16



Appendix J J MEAN SCORES FOR MEASURES CARD 5 - N GROUP

Administration	Moan f	or Measure	
Administration	5-1	.or Measure 5-2*	= 5-3**
	7-I	3-2"	3-3
First	1.02	1.00	. 47
Second	•93	• 99	.48
Third	.99	1.07	.48
Fourth	.97	1.00	• 50

^{* 5-2} is measured in inches
** 5-3 is measured in degrees



Appendix K K

MEAN AREAS FOR MEASURES

CARD 6 - N GROUP

Administration	Mean fo	r Measur	е	
	6-1	6–2	6-3	
First	1.31	.54	1.86	
Second	1.16	.58	1.74	
Third	1.55	.63	2.18	
Fourth	1.35	.59	1.90	



Appendix L L

MEAN AREAS FOR MEASURES

CARD 7 - N GROUP

Administration	Mean f	or Measu	re
	7-1	7–2	7–3
First	.84	.76	1.60
Second	.81	.72	1.53
Third	. 80	. 70	1.50
Fourth	.86	.76	1.62



Appendix M M

MEAN AREAS FOR MEASURES

CARD 8 - N GROUP

Administration	Mean for Measure	
***************************************	8-1 8-2 8-3	
First	1.33 .12 1.44	
Second	1.40 .12 1.53	
Third	1.38 .12 1.50	
Fourth	1.39 .12 1.51	



Appendix N N

MEAN AREAS FOR MEASURE

CARD 9 - N GROUP

Administration	Mean for Measure 9-1
First	10.73
Second	11.03
Third	11.46
Fourth	11.52



Appendix 0 0
ANALYSES OF VARIANCE

CARD A - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure A-1				
Treatments	.31	3	.105	3.27*
Blocks	1.85	24	.077	
Residual	2.31	72	.032	
TOTAL	4.47	99		
Measure A-2				
Treatments	.09	3	.031	2.81*
Blocks	1.16	24	.048	
Residual	.80	72	.011	
TOTAL	2.05	99		
Measure A-3				
Treatments	.74	3	.248	3.58*
Blocks	5.44	24	.227	
Residual	4.99	72	.069	
TOTAL	11.17	99		

^{*} p < .05



Appendix P P

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN LENGTHS FOR MEASURE 1-1

CARD 1 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Squares	F
Treatments	1.03	3	.346	.83
Blocks	65.11	24	2.713	
Residual	29.72	72	.412	
TOTAL	95.86	99		
	•			

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Appendix Q Q

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURE 2-1

CARD 2 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Treatments	1.16	3	.387	1.94
Blocks	36.15	24	1.506	
Residual	14.35	72	.199	
TOTAL	51.66	99		

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Appendix R R

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURE 3-1

CARD 3 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
				
Treatments	.19	3	.064	.94
Blocks	12.65	24	.527	
Residual	4.92	72	.068	
TOTAL	17.76	99		

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Appendix S S
ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 4 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 4-1				
Treatments	.14	3	.049	.177
Blocks	3.37	24	.140	
Residual	2.01	72	.027	
TOTAL	5.54	99		
Measure 4-2				
Treatments	.01	3	.005	.38
Blocks	1.49	24	.062	
Residual	. 97	72	.013	
TOTAL	2.47	99		
Measure 4-3				
Treatments	•00	3	.000	1.13
Blocks	.05	24	.002	
Residual	.04	72	.000	
TOTAL	.09	99		



Appendix T T

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES CARD 4 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 4-4				
Treatments	.00	3	.000	2.66
Blocks	.04	24	.001	
Residual	.03	72	.000	
TOTAL	.07	99		
,				
Measure 4-5				
Treatments	.32	3	.106	1.64
Blocks	7.94	24	.330	
Residual	4.69	72	.065	
TOTAL	12.95	99		

Appendix U U

ANALYSES OF VARIANCE

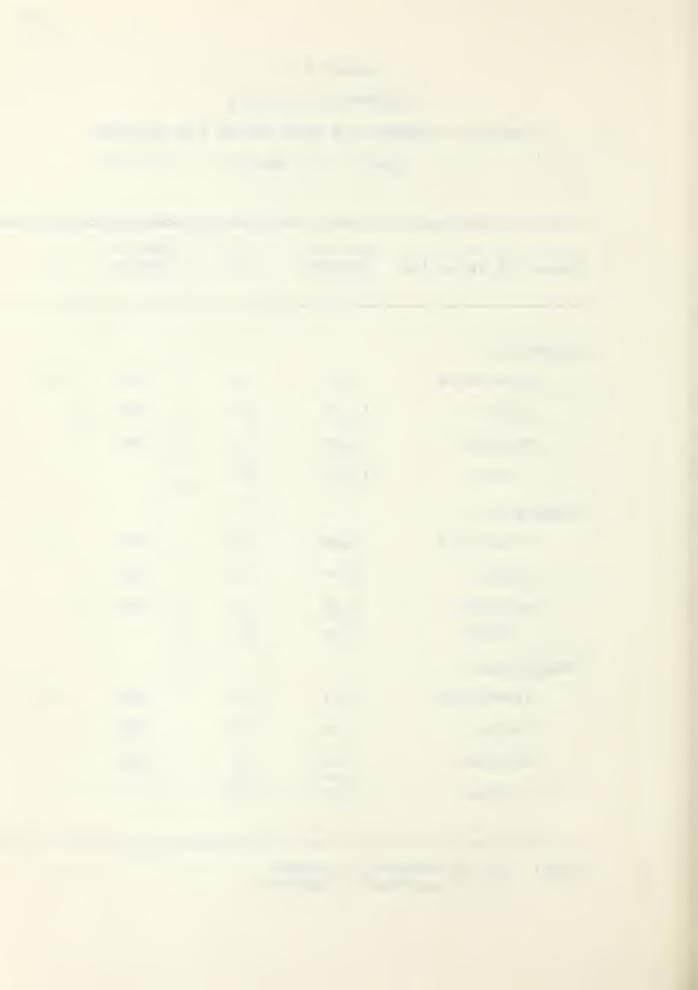
DIFFERENCE BETWEEN SIX MEAN SCORES FOR MEASURES

CARD 5 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 5-1				
Treatments	.11	3	.038	.68
Blocks	12.22	24	•509	
Residual	4.09	72	.056	
TOTAL	16.42	99	•000	
Measure 5-2				
Treatments	.08	3	.028	.73
Blocks	6.76	24	.281	
Residual TOTAL	2.80 9.64	72 99	.038	· .
Measure 5-3				
Treatments	.01	3	.004	. 98
Blocks	.80	24	.033	
Residual	.29	72	.004	
TOTAL	1.10	99		

Note: 5-2 is measured in inches

5-3 is measured in degrees



Appendix V V
ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 6 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 6-1				
Treatments	1.95	3	• 650	4.71*
Blocks	10.70	24	.445	** / 1
Residual		72	.138	
TOTAL	22.59	99		
Measure 6-2				
Treatments	.12	3	.040	1.56
Blocks	1.86	24	.077	
Residual	1.84	72	.025	
TOTAL	3.82	99		
Measure 6-3				
Treatments	2.68	3	.893	4.27*
Blocks	19.61	24	.817	
Residual	15.08	72	.209	
TOTAL	37.37	99		

^{*} p < .05



Appendix W W

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 7 - N GROUP

	Sum of		Mean	
Source of Variation	Squares	d.f.	Square	F
Measure 7-1				
Treatments	. 05	3	.018	.30
Blocks	5.78	24	.214	
Residual	4.40	72	.061	
TOTAL	10.23	99		
Measure 7-2				
Treatments	.06	3	.022	.69
Blocks	3.39	24	.141	
Residual	2.34	72	.032	
TOTAL	5.79	99		
Measure 7-3				
Treatments	.23	3	.079	.51
Blocks	16.58	24	.691	
Residual	11.17	72	.155	
TOTAL	27.98	99		

Appendix X X
ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURES

CARD 8 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Measure 8-1				
Treatments	.08	3	.027	.31
Blocks	12.22	24	.509	
Residual	6.46	72	.089	
TOTAL	18.76	99		
Measure 8-2				
Treatments	•00	3	.000	.34
Blocks	.16	24	.006	
Residual	•07	72	.001	
TOTAL	.23	99		
Measure 8-3				
Treatments	• 09	3	.032	.31
Blocks	14.27	24	.594	
Residual	7.45	72	.103	
TOTAL	21.81	99		



Appendix Y Y

ANALYSES OF VARIANCE

DIFFERENCE BETWEEN SIX MEAN AREA SCORES FOR MEASURE

9-1 - N GROUP

Source of Variation	Sum of Squares	d.f.	Mean Square	F
Treatments	10.58 -	3	3.527	1.94
Blocks	425.23	24	17.718	
Residual	131.24	72	1.822	
TOTAL	567.05	99		











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